CARDIOVASCULAR MEDICINE

Ethnic differences in the prevalence and aetiology of left ventricular systolic dysfunction in the community: the Harrow heart failure watch

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Objective: To assess ethnic differences in the prevalence and aetiology of left ventricular systolic dysfunction (LVSD) in the community.
Design: Community cohort study. All patients underwent echocardiography and those found to have LVSD underwent myocardial perfusion imaging with or without coronary angiography to diagnose underlying coronary artery disease (CAD).
Setting: Seven representative general practices in Harrow, UK, a community hospital, and a local district general hospital.
Patients: 1392 patients ⩾ 45 years old randomly selected from the computer records of seven general practices.
Main outcome measures: The prevalence and aetiology of LVSD in the community, assessing differences between white and non-white populations, and the proportion of patients with LVSD with undiagnosed CAD.
Results: 734 patients (53%) attended, 518 (71%) white and 216 (29%) non-white, the majority South Asian. Thirty nine patients (5.5%) had probable LVSD and 25 (3.5%) definite LVSD. No significant differences in prevalence were seen with ethnicity. CAD underlay most cases of LVSD. Non-white patients had a higher prevalence of CAD as the underlying aetiology of significant LVSD than white patients (100% v 56%, p = 0.04) and a trend towards less alcoholic cardiomyopathy. 8% of patients with LVSD had undiagnosed CAD.
Conclusions: LVSD is common. White and non-white patients have a similar overall prevalence of LVSD. Non-white patients, the majority South Asians in this study, have a higher prevalence of CAD as the underlying cause for LVSD than white patients. CAD underlies most cases of LVSD in the community, although it may be undiagnosed unless formally assessed.

Heart failure is one of the most common chronic disorders of the western world with high associated morbidity, mortality, and cost.[1] Left ventricular systolic dysfunction (LVSD) underlies most cases of heart failure, but is often asymptomatic before its development.[2] Intervention greatly reduces morbidity and mortality irrespective of symptoms.[3] A number of studies have evaluated the prevalence of LVSD in white populations, finding prevalences of about 2–5% in the general population[4–7] and higher in more elderly populations.[8 9] The one study that assessed a non-white population—that of American Indians, where diabetes mellitus is rife—found a much higher prevalence of LVSD of 14%, although the cut off used to define LVSD was higher than in the other studies.[10] No study has thus far assessed a multiethnic community directly comparing prevalences between ethnic groups.

Moreover, few data have been collected on the underlying aetiology of LVSD in the community, with no community study thus far formally testing for underlying coronary artery disease (CAD) in prevalent cases of LVSD. This is becoming increasingly important with the realisation that best medical treatment may differ between ischaemic and non-ischaemic LVSD and that revascularisation in patients with ischaemic LVSD and underlying hibernating myocardium may greatly improve prognosis.[11 12] The one community based study thus far that formally tested for underlying CAD in incident cases of heart failure found that an ischaemic aetiology was unknown in 13% of cases before formal testing.[13]

Accordingly, this study was undertaken to assess the prevalence and aetiology of LVSD in a multiethnic community, assessing differences with ethnicity. The population chosen, that of Harrow, North London, has a high prevalence of both white people and South Asians (people from the Indian subcontinent).[14] South Asians are known to have a high prevalence of diabetes mellitus and myocardial infarction and a higher risk lipid profile,[14] and thus may have a higher prevalence of LVSD and different underlying aetiologies than their white counterparts.

METHODS

We randomly selected 1403 people ⩾ 45 years of age from the computer records of seven geographically and socioeconomically representative general practices in Harrow, about 200 patients from each practice (range 199–202), from 48 practices. Of these patients, 11 were excluded, as they had died. Thus, 1392 patients were invited to attend. Patients were screened in a local community hospital and a local district general hospital. All patients were seen between January 2000 and May 2001.

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Abbreviations: CAD, coronary artery disease; CI, confidence interval; LVEF, left ventricular ejection fraction; LVSD, left ventricular systolic dysfunction; MIBI, methoxyisobutylisonitrile; SPECT, single photon emission computed tomography

Patient assessment

Attending patients filled in a questionnaire. Their height, weight, resting blood pressure (the average of two readings) and heart rate were measured. Spirometric measurements and ECG were recorded. Blood was drawn for fasting lipids, fasting glucose, creatinine, and natriuretic peptide analyses. The general practice computer records of attending and non-attending patients were compared.

Echocardiography

Two dimensional echocardiography was performed with a SONOS 4500 (Philips, Eindhoven, the Netherlands) using second harmonic imaging. Left ventricular ejection fraction (LVEF) was calculated quantitatively by Simpson's apical biplane rule,[15] the average of three consecutive normal cardiac cycles. Intraobserver and interobserver variabilities were assessed in 50 randomly selected cases independently by two observers (GG and RS) blinded to the initial result. Probable LVSD was defined as an ejection fraction below the 99th centile of LVEF calculated in attending patients who were free of potential risk factors for CAD and LVSD (hypertension, ischaemic heart disease, diabetes mellitus, cerebrovascular disease, peripheral vascular disease, and a history of heavy alcohol intake), or "normal" patients. Definite LVSD was defined as the cut off for probable LVSD in these normal patients minus the 95% limits of agreement for intraobserver variability.

Myocardial perfusion imaging

Patients found to have LVSD were invited to attend again for a stress–rest myocardial perfusion (methoxyisobutylisonitrile (MIBI)) scan within three months of their echocardiography study. For the stress study 600 MBq of technetium-99m sestamibi was injected intravenously after treadmill exercise if possible or after administration of 0.56 mg/kg dipyridamole if exercise was difficult. For the rest study, 600 MBq of ^{99m}Tc sestamibi was injected at rest after administration of 400 μg sublingual glyceryl trinitrate. Scintigraphic images were acquired 60 minutes after tracer injection with a double headed gamma camera (Sophy Medical, Buc Cedex, France). Images were divided into eight frames by ECG R wave triggering. Dynamic gated images were displayed by commercially available software (MyoSPECT, SMV, Ohio, USA). Gated single photon emission computed tomograms (SPECT) were analysed blinded to the echocardiography results by a single observer (AL). LVEF was calculated from the gated images by two validated commercially available software packages (QGS, Cedars-Sinai Health System, Los Angeles, California, USA and Emery Cardiac Toolbox, Syntermed, Atlanta, Georgia, USA).[16 17]

Coronary angiography

Patients with borderline abnormal or clearly abnormal MIBI scans suggestive of possible underlying CAD who were not known to have prior CAD (documented myocardial infarction, coronary artery bypass surgery, or abnormal coronary angiography) were invited to undergo coronary angiography for a formal diagnosis. Coronary angiography was performed with the Judkins technique. CAD was diagnosed as the underlying primary aetiology if a stenosis qualitatively graded as ⩾ 70% was seen in a major coronary artery or one of its major branches supplying akinetic or hypokinetic myocardial segments. Patients with completely normal MIBI scans did not go on to coronary angiography and were considered to have non-ischaemic LVSD, with assigned aetiology dependent on the questionnaire data, data from general practitioners, ECG findings, and echocardiography findings, leading to a partially subjective assessment.

Statistical analysis

Results from continuous data are given as mean (SD). Normally distributed continuous data were compared by Student's t test. Non-normally distributed data were compared by the Mann-Whitney U test. The Yates corrected or Fisher exact χ^2 test was used, where appropriate, to compare categorical groups. Binomial 95% confidence intervals (CI) were calculated for prevalences. Prevalences were defined as the number of people found to have LVSD divided by the number of people assessed. Data were analysed with Analyse-it for Microsoft Excel version 1.48 (Analyse-It Software Ltd, Leeds, UK).

The study was powered to detect a 5% excess in the prevalence of LVSD in South Asians over white patients with $\beta = 0.2$ and $\alpha = 0.05$, assuming a 50% response rate, that 95% of attending patients would be either South Asian or white in a ratio of 3:7, and that the overall prevalence of LVSD was 5%.

RESULTS

Patient demography

Table 1 lists the demographic characteristics of the 734 patients (53%) who attended. Table 2 shows demographic differences between attendees and non-attendees. There were 518 white (71%) and 216 (29%) non-white patients, 188 of whom (87%) were South Asians. Table 3 shows demographic differences with ethnicity. There were 444 normal patients.

Table 1 Demographic characteristics of study participants

Number seen	734
Age (years)	60 (10)
	(range 45–89)
Ethnicity	
White	518 (71%)
South Asian	188 (26%)
Other	28 (4%)
Men	349 (48%)
Hypertension	193 (26%)
Ischaemic heart disease	82 (11%)
Diabetes mellitus	45 (6%)
Cerebrovascular disease	24 (3%)
Peripheral vascular disease	9 (1%)
History of heavy alcohol intake (⩾40 U/week)	41 (6%)
Free of risk factors	444 (60%)

Data are mean (SD) or number (%).

Table 2 Demographic differences between attendees and non-attendees based on data from their general practice computer records

	Non-attendees	Attendees	p Value
Age (years)	63	60	<0.0001*
Men	50%	48%	0.49
Current smokers	12%	11%	0.58
Body mass index (kg/m²)	25.8	26.5	0.08
Alcohol intake (U/week)	5	4	0.34
Ischaemic heart disease	7%	10%	0.02*
Hypertension	23%	24%	0.53
Cerebrovascular disease	2.4%	2.2%	0.89
Diabetes mellitus	5.3%	5.2%	0.999
Peripheral vascular disease	0.3%	1.0%	0.19
Heart failure or loop diuretic usage	6.8%	4.5%	0.07
Any risk factor†	34%	36%	0.47

Data are mean or percentage.
*Significant difference; †any of angina, myocardial infarction, heart failure or loop diuretic usage, hypertension, diabetes mellitus, cerebrovascular disease, or peripheral vascular disease.

Table 3 Demographic differences by ethnicity

	White	Non-white	South Asian	p Value (South Asian v white)
Number seen	518	216	188	NA
Age (years)	60.5 (11) (range 45–89)	57.2 (9) (range 45–78)	57.0 (9) (range 45–78)	0.0002*
Men	48%	45%	45%	0.49
Body mass index (kg/m^2)	26.1 (4.1)	26.0 (4.2)	26.0 (4.2)	0.86
HDL cholesterol (mmol/l)	1.48 (0.4)	1.38 (0.4)	1.37 (0.3)	0.0009*
Total cholesterol:HDL	4.0 (1.2)	4.0 (1.1)	4.0 (1.1)	0.94
Hypertension	25%	29%	29%	0.41
Myocardial infarction	4.8%	6.5%	6.4%	0.40
Ischaemic heart disease	11%	12%	12%	0.74
Diabetes mellitus	4%	11%	11%	0.0008*
Cerebrovascular disease	3%	3%	3%	0.96
Heavy drinker (≥40 U/week)	7%	2%	2%	0.02*
Peripheral vascular disease	1.5%	0.5%	0.5%	0.45
No medical risk factors	61%	60%	60%	0.93

Data are man (SD) or percentage.
*Significant difference.
HDL, high density lipoprotein.

Interobserver and intraobserver variabilities in LVEF

Intraobserver variability gave a mean overall difference in LVEF of 0.1% and 95% limits of agreement of ±6%. Interobserver variability gave a mean overall difference of 1.3% and 95% limits of agreement of ±8%.

Echocardiography

LVEF was calculable in 706 cases (96%). The mean LVEF in the 444 normal patients free of risk factors was 61.7 (4.7)% (fig 1). The 99th centile of normality was 50.7%. Probable LVSD was taken as LVEF < 50% (the 99.3rd centile of normality) and definite LVSD as LVEF < 45% (the 99.98th centile of normality). There were no significant differences in LVEF between white and non-white patients free of risk factors (61.7 (4.9)% v 61.7 (4.3)%, respectively, p = 0.89).

Prevalence of LVSD

Thirty nine patients (5.5%, 95% CI 4.0% to 7.5%) had probable LVSD, 9.0% of men and 2.4% of women (p = 0.0002). Of these, 18 (46%) were entirely asymptomatic and not taking loop diuretics; only 12 (31%) had a general practitioner's diagnosis of heart failure or LVSD or were prescribed loop diuretics, and only 12 (31%) were taking disease modifying medication (angiotensin converting enzyme inhibitors, angiotensin II antagonists, aldosterone antagonists, or disease modifying β blockers). Twenty five patients (3.5%, 95% CI 2.3% to 5.2%) had definite LVSD, 6.0% of men and 1.3% of women (p = 0.002). Of these, eight

Figure 1 Frequency distribution of left ventricular ejection fraction in 444 patients free of risk factors for left ventricular systolic dysfunction and coronary artery disease.

(32%) were entirely asymptomatic and not taking loop diuretics; only nine (36%) had a general practitioner's diagnosis of heart failure or LVSD or were prescribed loop diuretics and only 10 (40%) were taking disease modifying medication. The prevalence of LVSD increased with age (p < 0.001, χ^2 test for trend) (table 4) and was greater in men than women in each age group. In the analysis of increased severity of LVSD, 15 patients (2.1%, 95% CI 1.2% to 3.5%) were found to have LVEF < 40% and 11 patients (1.6%, 95% CI 0.8% to 2.8%) were found to have LVEF < 35%.

No significant differences in prevalence were seen among ethnic groups. White patients had a prevalence of probable LVSD of 5.6% (95% CI 3.8% to 8.0%), non-white patients of 5.3% (95% CI 2.7% to 9.3%, p = 0.997 v white patients), and South Asians of 5.0% (95% CI 2.3% to 9.3%, p = 0.89 v white patients). White patients had a prevalence of definite LVSD of 3.6% (95% CI 2.2% to 5.7%), non-white patients of 3.4% (95% CI 1.4% to 6.8%, p = 0.95 v white patients), and South Asians of 3.3% (95% CI 1.2% to 7.1%, p = 0.95 v white patients). Table 5 shows the age and sex adjusted prevalences of LVSD in white patients and South Asians, which were not significantly different.

Aetiology of LVSD

Thirty three patients with LVSD underwent myocardial perfusion imaging and 10 underwent coronary angiography. Eight patients underwent both tests and four patients neither test. After myocardial perfusion imaging and angiography, three of the 39 patients with borderline LVSD (8%, 95% CI 2% to 21%) and one of the 25 patients with definite LVSD (4%, 95% CI 0% to 20%) were found to have unknown significant CAD.

The final underlying primary aetiologies for probable LVSD were CAD in 56%; alcoholic cardiomyopathy in 13%; hypertensive cardiomyopathy in 8%; diabetes mellitus in 5%; valvar heart disease in 5%; and unknown in 13%. The final primary aetiologies for definite LVSD were CAD in 68%; alcoholic cardiomyopathy in 12%; hypertensive cardiomyopathy in 8%; valvar heart disease in 8%; and unknown in 4%.

Table 6 shows the prevalences of the primary aetiological factors underlying LVSD within each ethnic group. Seventy three per cent of non-white patients versus 50% of white patients had underlying CAD as the primary aetiology of probable LVSD (p = 0.18). Eighteen per cent of white patients versus 0% of non-white patients had alcoholic cardiomyopathy as the primary aetiology of probable LVSD
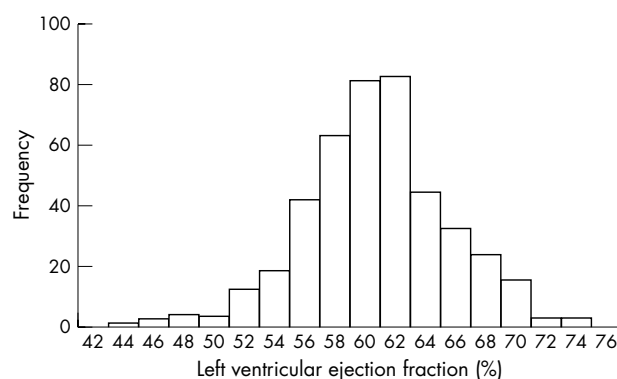
Table 4 Prevalence of probable (LVEF <50%) and definite left ventricular systolic dysfunction (LVEF <45%) stratified by age and sex

		Age group (years)				
		45–54	55–64	65–74	75–84	≥75
LVEF <50%	Men	5.0%	6.6%	12.0%	20.0%	21.1%
	Women	0.0%	4.9%	0.0%	2.9%	7.1%
	All	2.2%	5.6%	7.0%	11.4%	13.7%
LVEF <45%	Men	2.5%	3.3%	10.8%	11.4%	13.2%
	Women	0.0%	1.6%	0.0%	2.9%	7.1%
	All	1.1%	2.3%	6.3%	7.1%	10.0%

LVEF, left ventricular ejection fraction.

(p = 0.17). One hundred per cent of non-white patients versus 56% of white patients had underlying CAD as the primary aetiology of definite LVSD (p = 0.04). Seventeen per cent of white patients versus 0% of non-white patients had alcoholic cardiomyopathy as the primary aetiology of definite LVSD (p = 0.35).

Comparison of echocardiography with gated SPECT LVEF
Thirty three patients had LVEF assessed by gated SPECT and by echocardiography, all with LVEF < 50% on echocardiography. QGS software gave a mean LVEF 2.2% lower than with echocardiography (39.1% v 41.2%, p = 0.08). Emory Cardiac Toolbox gave a mean LVEF 0.4% lower than with echocardiography (40.8% v 41.2%, p = 0.72). None of these differences reached significance.

DISCUSSION
This is the first community based study to evaluate ethnic differences in the prevalence and aetiology of LVSD in the community. We found no significant differences in the prevalence of LVSD but significant differences in the underlying aetiology between ethnic groups. Our results have further helped validate the use of echocardiography in screening for LVSD in the community, a role recently questioned in comparative studies,[18 19] finding tight interobserver and intraobserver variabilities and no significant difference between echocardiography and gated myocardial SPECT imaging in patients who were assessed.

What is the normal range of LVEF in the community?
This study has gone on to help define the normal range of LVEF in the community. By using second harmonic imaging and analysing digital data from loop recordings, we were able to calculate LVEF in 96% of patients, with tight intraobserver and interobserver variabilities. This was an improvement from two earlier studies that used fundamental imaging and videotape analysis, in which LVEF was calculable in only 89.5% and 65% of cases, respectively.[4 9] We found a mean LVEF of 62% in the healthy population free of cardiac risk

factors and found no significant differences with ethnicity. We further found LVEF < 50% as the 99.3rd centile of normality and probable abnormality and LVEF < 45% as the 99.98th centile of normality and definite abnormality. No significant differences were seen between echocardiography and gated SPECT perfusion imaging in patients assessed by both techniques, further validating these results. These cut offs are in keeping with other epidemiological studies and published guidelines,[5 20 21] with treatment benefit seen in symptomatic patients with LVEF < 45% undergoing treatment.[22 23]

Prevalence of LVSD in the general population
This study has provided further evidence of the high prevalence of LVSD in the general population, finding an overall prevalence of definite LVSD of 3.5% and probable LVSD of 5.5%. It has found an increase in prevalence with age and with male sex. Almost half of affected patients were symptom-free, with two thirds having undiagnosed heart failure or LVSD and not taking disease modifying medication. Similar results have been seen in purely white populations. McDonagh et al[4] found a prevalence of definite LVSD of 2.9% and of probable LVSD of 7.7%; Davies et al[5] found a prevalence of definite LVSD of 1.8% and of probable LVSD of 5.3%; Mosterd et al[6] found a prevalence of LVSD of 3.7%; and Schunkert et al[7] found a prevalence of LVSD of 2.7%. All four studies also found an increase in prevalence with age and male sex. The one study thus far that assessed a non-white population, that of American Indians, found a higher prevalence of possible LVSD of 14% (defined as LVEF < 54%) but a similar prevalence of definite LVSD of 2.9% (defined as LVEF < 40%), also finding an increase with male sex and increasing age.[10] This study has helped assess whether such differences with ethnicity exist.

Differences with ethnicity
This study found no differences in the overall prevalence of LVSD between white and non-white patients, the majority being South Asian in this study. However, we did find significant differences in the underlying aetiology of LVSD, finding a higher proportion of underlying CAD in non-white patients. Thus, although there was a higher prevalence of ischaemic cardiomyopathy in non-white patients than in white patients (3.4% v 2.0%, respectively, for LVEF < 45%; and 3.8% v 2.8%, respectively, for LVEF < 50%), there was no overall excess of LVSD in non-white patients due to fewer cases of non-ischaemic cardiomyopathy.

Potential risk factors for CAD and thus ischaemic cardiomyopathy in South Asians include a lower concentration of high density lipoprotein cholesterol and higher prevalence of diabetes mellitus as seen in this and other studies.[14] Other potential factors are genetic factors, with a greater tendency to central obesity in South Asian children compared with white children,[24] and a higher prevalence of insulin resistance, independent of diabetes mellitus.[25]

Table 5 Age and sex adjusted prevalences of probable (LVEF <50%) and definite left ventricular systolic dysfunction (LVEF <45%)

		White	South Asian	p Value
LVEF <50%	Men	8.4%	6.3%	0.74
	Women	1.0%	4.0%	0.13
	All	4.3%	5.0%	0.83
LVEF <45%	Men	5.4%	6.3%	0.78
	Women	0.4%	1.0%	0.50
	All	2.6%	3.3%	0.60

Table 6 Prevalence of primary aetiological factors underlying probable (LVEF <50%) and definite left ventricular systolic dysfunction (LVEF <45%) stratified by ethnic group

Primary aetiological factor	White		Non-white		South Asian	
	LVEF <50%	LVEF <45%	LVEF <50%	LVEF <45%	LVEF <50%	LVEF <45%
Coronary artery disease	50%	56%	73%	100%	78%	100%
Alcoholic cardiomyopathy	18%	17%	0%	0%	0%	0%
Hypertension	7%	11%	9%	0%	0%	0%
Diabetes mellitus	4%	0%	9%	0%	11%	0%
Valvar heart disease	7%	11%	0%	0%	0%	0%
Unknown	14%	6%	9%	0%	11%	0%

Certainly, South Asians have been shown to develop myocardial infarctions[14] and heart failure[26] at a younger age than white patients and have more extensive myocardial infarctions than their white counterparts.[14]

One factor underlying the lower prevalence of non-ischaemic cardiomyopathy in South Asians was a lower rate of alcoholic cardiomyopathy. This was in turn due to a significantly lower frequency of heavy alcohol intake found among South Asians in the baseline demographic data. McKeigue and Karmi[27] found a similar very low prevalence of heavy alcohol intake among South Asians as compared with the native British population and a significantly higher rate of alcohol abstinence among South Asians, confirming this further.

Although this is the first community based study to evaluate ethnic differences in the prevalence and aetiology of LVSD in the community, some prior studies have evaluated the ethnicity of patients presenting to hospital in heart failure. Lip et al[28] found that 16% of patients presenting to a hospital in Birmingham were of South Asian ethnicity, with South Asians generally presenting at a younger age than their white counterparts. Although 25% of adults in that local population were of South Asian ethnicity, suggesting a lower overall hospitalisation rate for South Asians, a reanalysis of these data in relation to census estimates found that elderly South Asians aged between 60 and 79 years had a fivefold higher risk of presenting with heart failure than their white counterparts.[29] Similarly, Blackledge et al[26] found that South Asians had higher age adjusted admission rates for heart failure in Leicestershire than white patients, again generally presenting at a younger age. Neither study could, however, fully exclude ethnic specific biases in referral rates or in diagnosing heart failure.

Combining the findings of these earlier studies with that of the current study appears to suggest that, although South Asians have a similar prevalence of LVSD to white patients, they have a higher rate of hospitalisation for heart failure. This may result from South Asians having more severe heart failure, once developed, or alternatively more diastolic heart failure (heart failure with normal LVEF). Both mechanisms appear likely. South Asians with heart failure are more likely to have hypertension, diabetes mellitus, and CAD than white patients[26] and to have had more extensive myocardial infarctions.[14] These extra co-morbidities may both underlie an increased likelihood of heart failure hospitalisation and be risk factors in the development of diastolic heart failure.[26] Future studies are required to evaluate this further.

Aetiology of LVSD
Although this is the first study to assess the prevalence of CAD accurately in prevalent cases of LVSD in the community, Fox et al[13] assessed the prevalence of underlying CAD in incident cases of heart failure, finding similar results. The proportion of cases of LVSD attributable to CAD increased from 39% to 52% after coronary angiography. This compares with an increase from 49% to 56% in the current study.

Four community based studies found similar low rates of underlying CAD in patients with prevalent heart failure when not formally testing for underlying CAD, finding rates between 32% and 45%.[30–33] It is likely that if patients had been tested formally then this proportion would have increased as in the current study. Indeed, a fifth to a third of cases of myocardial infarction are known to go unrecognised, especially among elderly and diabetic patients,[34 35] who are themselves at higher risk of developing LVSD. This may explain the higher prevalence of underlying CAD in hospital based heart failure studies, where underlying aetiology is likely to be more stringently assessed, with a mean prevalence of 68% in one large meta-analysis of over 20 000 patients with heart failure.[36] Differentiating patients with underlying ischaemic heart disease may be important, as patients with ischaemic cardiomyopathy have a worse prognosis than those with non-ischaemic cardiomyopathy, respond differently to treatment, and may receive benefit from revascularisation.[11 12]

Study limitations
One potential study limitation is the relatively low response rate of 53%, giving the potential of a non-response bias potentially affecting the generalisability of the study. However, although there were some demographic differences between attendees and non-attendees, no significant difference in overall risk factors for LVSD were seen, making this less likely. Similar response rates have also been seen in other similar studies, with Redfield et al[37] finding a response rate of only 47% and McDonagh et al[4] an overall response rate of only 56%. Davies et al,[5] in the only such study to perform echocardiography at general practitioner surgeries rather than in hospital, had a higher response rate of 63%. Thus, although some authors have suggested that the general population should be screened for asymptomatic LVSD,[2] these low response rates make hospital based screening unlikely to be workable. A more attractive option, currently under discussion, would be that of community based screening for LVSD with the newer screening modalities of hand held echocardiography or natriuretic peptide blood tests, methods ideally suited to community screening.[38 39] Secondly, it was not possible to ascertain the ethnic group or socioeconomic status of invitees, only of attendees; thus, differences in response rate between ethnic groups and socioeconomic class could not be assessed. Any systematic difference in response rate between ethnic groups or within socioeconomic class may affect the generalisability of the study to the community as a whole. Finally, although no significant difference in the prevalence of LVSD was seen between white patients and South Asians, the study was only powered to detect a difference in prevalence of 5% between these two ethnic groups. It is possible that a smaller but still

significant difference exists between these groups, with a much larger study required to answer this further.

Conclusion

LVSD is a common condition, with prevalence increasing with male sex and increasing age. Two thirds of patients with LVSD had undiagnosed heart failure or LVSD and were not taking disease modifying medication. No differences in the prevalence of LVSD were seen between white and non-white patients. Non-white patients, the majority being South Asians in this study, had a significantly higher prevalence of underlying CAD and a trend towards less alcoholic cardiomyopathy as the underlying cause of LVSD. CAD underlies most cases of LVSD, although it may be undiagnosed in 8% of cases.

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Authors' contributions: Gavin Galasko helped design the study, performed most of the echocardiography, analysed and interpreted the data, and wrote the paper. Roxy Senior helped design the study, supervised the echocardiography, and contributed to the writing of the paper. Avijit Lahiri had the initial idea for the study, helped design it, analysed the myocardial perfusion imaging results, and contributed to the writing of the paper.

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